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MAINTAINING THE SOMATO-FUNCTIONAL STATUS, A DETERMINING FACTOR IN STIMULATING NEUROPLASTICITY PROCESSES IN PATIENTS WITH NEUROGENIC DISORDERS.

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Abstract: The presented study is based on an analysis of the pathophysiological factors that are found in the clinical picture of neurogenic disorders that produce paralysis and implicitly the installation of deconditioning mechanisms in the organism. Paralysis can have various manifestations and negative effects on the body's function by limiting metabolic physiological processes. The age of the neurogenic patient, the genetic background, the type of disease, its complexity and possible comorbidities, condition and direct the development of the therapeutic path. One of the main goals in the clinical management of these patients is to maintain the neuro-vasculo-trophic status at physiologic parameters and to limit the negative effects of neurogenic conflict. Therefore, we will try to realize a model of recuperative therapeutic conduct in neurogenic disorders starting from the positive diagnosis and clinico-functional assessment.

Introduction:

In order to understand the mechanisms of deconditioning of the somato-functional status following the onset of a neurogenic disorder, we will present some elements of the anatomophysiology of the nervous system.

The nervous system receives, transmits and integrates information from the external and internal environment, on the basis of which it develops appropriate motor and secretory responses. Through the reflex function underlying its activity, the nervous system, together with the endocrine system, contributes to the functional unity of the organism and the dynamic balance between the organism and its environment. Nerve tissue is made up of neurons, the specifically differentiated cells that generate and conduct nerve impulses, and glial cells that form the supporting or interstitial tissue of the nervous system. The organs of the CNS - the nevraxus - are the spinal cord, brain stem, cerebellum, diencephalon and cerebral hemispheres. Virtually all of the body's activities are regulated by a nerve network of connecting nerve fibers, which branch off from the central nervous system and form the peripheral nervous system. [7, 9]

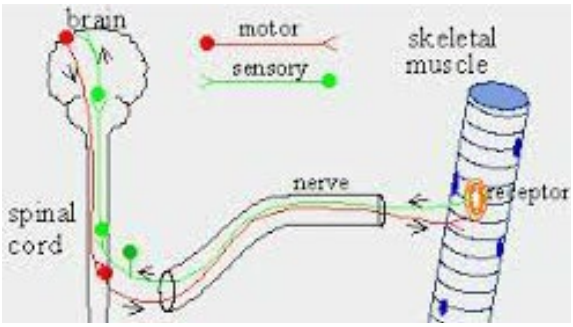

Fig.1 neuronal circulation pattern (diference) [14]

The reflex act is the physiological process of responding to a stimulus acting on a specific receptor field and has as its anatomical substrate the reflex arc, consisting of: the afferent pathway, a center and the efferent pathway. The afferent pathway is represented by the receptor and the nerve fibers corresponding to the reflex center. Receptors are specialized cell formations or dendrites of neurons in spinal ganglia or their cranial counterparts. Reflex centers are nerve formations, at the level of which the information collected by the receptors reaches and is processed. In nerve centers located at different levels, impulses are generated that reach the effector organs. Reflex or voluntary movement requires the integrity of the afferent and efferent pathways, cortical and subcortical nerve centers, and the muscular effector.

Receptors are differentiated formations for the detection and reception of energy variations, from outside or inside the organism, and their transformation into nerve impulses. Depending on their localization, they are classified into proprioceptors, extrareceptors and interoceptors.

Proprioceptors (kinesthetic receptors) are found in skeletal muscles, tendons, joints, labyrinth and are involved in the regulation of motor functions. They also belong exclusively to the class of mechanoreceptors, which signal the velocity, tension and shortening of muscles: muscle receptors: neuromuscular spindles and Golgi tendon Golgi; joint receptors: Ruffini, Golgi- Mazzoni and Vater- Pacini corpuscles. [11]

The recovery from traumatic brain injuries, strokes, tumors, degenerative diseases such as Parkison's disease or progressive diseases such as multiple sclerosis are increasingly being researched and the literature is quite extensive.

Aim and objectives of the study:

Aim of the work: argumentation of the analysis of the pathophysiological factors that are found in the clinical status of neurogenic disorders that produce paralysis and implicitly the installation of deconditioning mechanisms in the organism.

Research objectives:

- analysis of the specialized literature regarding of the neurorehabilitation therapy after paralysis;
- identifying and understanding the pathophysiological mechanisms leading to deconditioning of somatofunctional status;
- the realization of a therapeutic pathway favoring neuroplasticity processes.

Materials and methods: Within the neurogenic pathologies that cause paralysis and implicitly deconditioning of the somato-functional status, we will present some aspects of pathophysiology that confirm the need to establish a program to preserve function in a timely intervention. Transection of the motor nerve of the muscle can cause paralysis, i.e. complete loss of motility and reflex contractions. [1] The rehabilitation of a neurogenic patient depends on the nature of the etiologic factor, the complexity and extent of the conflict and the occurrence of complications. Prolonged immobilization in bed due to motor deficit paralysis can lead to cutaneous trophic disorders, pulmonary complications, deep vein thrombophlebitis and urinary tract infection.[11] Late complications occur within 4-6 weeks and present a complex clinical pattern: cardio-vascular, digestive, renal, locomotor, and locomotor system disorders, in addition to peripheral nervous, sexual (vegetative) and psychic disorders.[11]

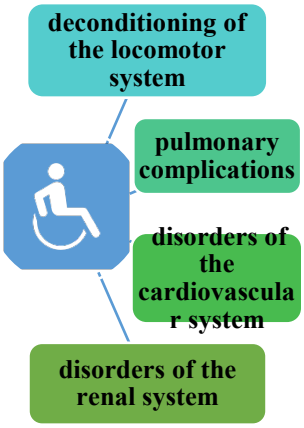

Fig.2 complications of reduced motor activity due to paralysis

One of the major objectives of the rehabilitation program is to maintain the neuro-vascular-trophic status of the locomotor system. This is necessary due to the deconditioning that is taking place at its level (musculature, joints and bones). Due to prolonged immobilization, the muscles become significantly amyotrophic. Musculo-tendinous retractions are more serious, due to vicious positions. Osteoporosis and, less frequently, para-osteoarthropathies also occur in bone tissue. [11] Also in this context, we will specify a particularly important aspect, namely that most of the deconditioning processes that are installed as an effect of immobilization, have an evolutive and irreversible trend with a tendency to structure in a vicious way. With regard to the neuroplasticity mechanisms accompanying the recovery process following a neurogenic disorder, a punctual analysis of all etiopathogenic and pathophysiological factors is required in order to stabilize the neuro-vascular-trophic stsatus and limiting the deconditioning processes installed.

The term "neuroplasticity" is used to describe the ability of neurons and neuronal aggregates to adjust their activity and even their morphology to changes in their environment or in their utilization patterns. The term also encompasses diverse processes, from learning and memory in the execution of normal activities of life, to dendritic cutting and axonal sprouting in response to injury.

Also in the same context, the term "neural repair", which has been introduced in the last few years to describe the range of interventions by which neural circuits lost as a result of injury or disease can be restored. Neuroplasticity, also known as neural plasticity or brain plasticity, is a process that involves adaptive structural and functional changes to the brain. [10]

This term includes means of enhancing axonal regeneration, transplantation of a variety of tissues and cells to replace lost neurons, and the use of prosthetic neural circuits to connect parts of the nervous system that have become functionally separated by injury or disease. Although there is some overlap with aspects of "neuroplasticity," the term "neuronal repair" generally refers to processes that do not occur spontaneously in humans at a level sufficient to lead to functional recovery. Therefore, therapeutic intervention is required to promote repair. The term is useful as part of the basic science of neurorehabilitation because it encompasses more than "regeneration" or "transplantation" alone. In recent years, the concepts of neural plasticity neural plasticity have been accepted as important elements in the scientific understanding of functional recovery. [6]

The term “neuronal plasticity” was already used by the “father of neuroscience” Santiago Ramón y Cajal (1852-1934) who described nonpathological changes in the structure of adult brains. [3]

The most appealing phenomenon of neuroplasticity appears to be adult neurogenesis, that is the generation of new neurons in adult brains. [3]

As a concept, neuroplasticity means the nervous system's ability to reorganize its structure and functioning in response to some stimuli, either intrinsic or extrinsic.[2]

Broadly speaking, neuroplasticity refers to the ability of the brain to undergo morphological and neurochemical changes as a result of experience.[12]

The human brain is composed of approximately 100 billion neurons. Early researchers believed that neurogenesis, or the creation of new neurons, stopped shortly after birth. Today, it's understood that the brain's neuroplasticity allows it to reorganize pathways, create new connections, and, in some cases, even create new neurons. [13]

Dr. David C. Kidd explains the process of neuroplasticity as the ability of the central nervous system to adapt and reorganize itself in response to experiences, learning and activity. This process can be described through four stages:

- Molecular changes: First of all, experience and activity create molecular changes in the brain, such as increased or decreased levels of neurotransmitters and proteins. These molecular changes underlie subsequent neuronal changes;
- Neuronal changes: Molecular changes then trigger neuronal changes, such as changes in dendrites and axons. This process can also include increasing the number of synapses between neurons, or adjusting existing connections;
- Neuronal circuits: neuronal changes can lead to changes in neuronal circuits, such as the formation of new neural pathways or changes in the way existing neural circuits function;
- Behavior: Finally, changes in neural circuitry can lead to changes in a person's behavior. For example, if a person practices a particular activity for a long time, such as playing the piano, the relevant neural connections may strengthen or new connections may form, allowing better performance.

Essentially, neuroplasticity is the process by which our experiences and activities can change the neuronal connections in the brain, adapting it to new demands and experiences. This process can be influenced by various activities, such as meditation, sports or cognitive games, suggesting that the ability to change the brain is not limited to the developmental period, but can be continuous into adult life. Gene expression, neurotrophic factors, axonal transport, collateral sprouting and neurogenesis are all factors that influence plasticity in the human brain. [5, 8]

All these factors are interdependent and may influence plasticity differently depending on the cortical region and neuronal function affected. Overall, however, these factors can help improve plasticity and recovery from injury in the human brain. Although it was previously thought that glial cells have a more passive and

supportive role in brain function, recent research has shown that they play a crucial role in brain plasticity and injury repair. In the case of injury, glial cells play an important role in nerve tissue repair. [4]

The plasticity of neuronal networks can be realized at the nerve endings of neurons that are continually degenerating while new ones are being formed.[1]

Results and discussions: In order to support the arguments presented above regarding the understanding of neuroplasticity mechanisms that may develop in the covalescence period after a neurogenic pathological status, we will present some aspects necessary for the elaboration of a therapeutic neurorehabilitation behavior. Any clinical reasoning starts from a positive diagnosis accompanied by a history and current investigations of the patient. Establishing the functional rest and stabilization of the patient are essential elements in order to develop a therapeutic plan that supports and promotes neuroplasticity processes. In table no.1 we present some conditions that the neurogenic patient must realize in order to sustain and develop neuroplasticity processes.

Table no.1 conditions for boosting neuroplasticity status

anatomical integrity	*surgery*
	hemodynamic stabilization
	isolation/elimination neurogenic conflict
stimulating trophicity	*vascular circulation*
	nervous circulation
	lymphatic circulation
neurorehabilitation	*postures/mobilizations to maintain muscle tone and joint mobility*
	physiotherapy

Conclusions:

- The main factors that condition and direct the development of the therapeutic pathway are: the neurogenic patient's age, genetic background, type of disorder, its complexity and possible

comorbiditiesclinical and paraclinical investigations determine the therapeutic course of lumbar disc disease;

- the main objective in the clinical management of neurogenic patients is to maintain the neuro-vasculo-trophic status at physiologic parameters and to limit the negative effects of the pathologic conflict;
- The plasticity of neural networks can be realized at the nerve endings of neurons that are continually degenerating while new ones are being formed.

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